Doctor Nelson's conclusions were based on a study of several local epidemics of flatulent diarrhea in infants and adults. Hanging-drop preparations of liquid stools revealed many encapsulated diplobacilli, which were very active gas producers. "After fifteen to twenty minutes' incubation, microscopic globules of gas were seen gathering about the periphery of each diplobacillus. In the course of a half-hour's incubation, the bubbles were escaping from each bacillus much more rapidly than could be counted." By routine cultural methods this gas producer was identified as a spore-forming anaerobe closely resembling *Cl. welchii*. Examination of the pasteurized milks associated with these cases revealed the presence of the same gas-producing anaerobe.

Under the belief that the growth of intestinal anaerobes is normally inhibited by the presence of acid-producing bacteria, Doctor Webster's clinical associates placed these cases on a sour milk ("culture milk") diet. The diarrhea and flatulence are alleged to have yielded at once to this diet. In a few cases recurrence followed a return to normal diet, but subsided on resuming the use of sour milk.

While Doctor Nelson's clinical conclusions are open to several interpretations, he has rendered a valuable service to clinical science by dramatizing the fact that, thus far, insufficient attention has been given to the heat-resistant spores in contaminated milks. Many of these spores survive and may actually profit by pasteurization, routine heating destroying the inhibiting acidifiers, driving out much of the dissolved oxygen, and inactivating lactic antibodies.

Stanford University.

W. H. MANWARING,
Palo Alto.

THE NATURE OF ANESTHETIC SHOCK AND THE VALUE OF PREMEDICATION

It is well established that reduction in the volume of circulating blood is the centrally important factor in shock. Recently Freeman [1] has emphasized the fact that many influences known to produce shock, or to aggravate the condition if present, are also known to be adequate stimuli for producing hyperactivity of the sympathetic nervous system. Thus vasoconstriction or increased output of adrenin, both of which are indices of generalized sympathetic hyperactivity, are caused by hemorrhage, cold, fear, pain, asphyxia, infection, and low blood pressure. It has been shown directly that vasoconstriction and increased output of adrenin are present in shock. Now Freeman has shown that prolonged hyperactivity of the sympathetic system as produced by continuous injection of epinephrin, or as present in the pseudaffective state following decortication, causes a decrease in the volume of the circulating blood. This effect is prevented by ergotoxin paralysis of the sympathetic, and in the pseudaffective state by complete sympathectomy.

That ether anesthesia has an effect on blood volume was shown by Barbour,[2] who demonstrated concentration of the blood. That ether affects the sympathetic nervous system is a recent conclusion by Bhatia and Burn,[3] who, following Elliott's early demonstration [4] that the adrenin content of the adrenals is reduced by ether and chloroform anesthesia, have shown that these anesthetics produce widespread stimulation of the sympathetic nervous system.

Thus the conclusion is evident that the secondary or delayed shock that may be produced by prolonged anesthesia with ether or chloroform is due to a concentration and reduction in volume of the circulating blood resulting from general stimulation of the sympathetic nervous system, including the outpouring of adrenin.

There has been an increasing amount of evidence that a number of drugs used as basal anesthetics or hypnotics, such as amytal, avertin, have a pronounced effect on the autonomic nervous system, centrally and peripherally. Particularly has the effect of amytal been studied on the experimental production of hyperglycemia. The administration of amytal is known to inhibit the hyperglycemia ordinarily brought about by asphyxia and by morphin,[5] presumably by preventing the reflex liberation of adrenin from the adrenals. Banerji and Reid [6] have just shown that in the hyperglycemia caused by ether and chloroform, the adrenals are importantly concerned, and have again demonstrated that such production of hyperglycemia is inhibited by amytal.

Does amytal, with related substances, prevent the general hyperactivity of the sympathetic nervous system that ether and chloroform produce? If they do, and there is some such evidence for such a conclusion, then their use as basal narcotics receives important support in that the tendency of such volatile anesthetics to produce shock may be markedly counteracted.

Pharmacological Laboratory,
University of California.

P. K. KNOEFEL,
San Francisco.

THE MECHANISM OF TRAUMATIC SHOCK

The mechanism of traumatic shock is being subjected to renewed scrutiny. The recent interest in this perennially important subject is due to the obvious analogy between shock and adrenal cortical insufficiency. In both, the loss of fluid from the vascular system seems to be the underlying cause of symptomatology. The classically accepted theory that the production of histamin in the tissues by trauma increases capillary permeability, permitting the escape of fluid from the vascular

1 Freeman, N. E.: Amer. Jour. Physiol., 103:185, 1933.

2 Barbour, H. G., and Bourne, W.: Amer. Jour. Physiol., 67:399, 1924.

3 Bhatia, B. B., and Burn, J. H.: Jour. Physiol., 78:257, 1933.

4 Elliott, T. R.: Jour. Physiol., 44:374, 1912.

5 Evans, C. L., Tsai, T., and Young, F. G.: Jour. Physiol., 72:1931. Olmstead, J. M. D., and Giragossintz, G.: Jour. Lab. and Clin. Med., 16:354, 1931. Brill, S.: Proc. Soc. Exper. Biol. Med., 27:265, 1930.

6 Banerji, H., and Reid, C.: Jour. Physiol., 78:370, 1933.